lytic myofibers (IIb + IIx), and an overall decrease in capillary density was observed in CSQ/CHF mice compared to WT littermates. Analysis of fiber type-specific capillary density revealed an increase in type IIa myofiber capillary density and a concomitant decrease in type IIb + IIx myofiber capillary density in the CSQ/CHF mice. **Conclusion**. These results suggest that chronic heart failure causes skeletal muscle fiber type switching and dysregulation of angiogenesis in a fiber type-specific manner. Furthermore, these results support the use of the CSQ/CHF mouse model to further study the role of skeletal muscle maladaptation in chronic heart failure and to explore potential therapeutic targets.

Fiber Type-Specific Angiogenic Dysregulation

	WT	CHF
Fiber Type	%	%
I	6.8	0.6
IIa	56.1	39.9
IIb+IIx	37.1	59.5
Capillary Density	Fibers/Cap	Fibers/Cap
IIa	1.66	2.43
IIb+IIx	0.73	0.47

1045-197 **Evidence for Hypoxia and Hypoxia-Inducible Factor-1-Alpha Mediated Vascular Endothelial Growth Factor Expression in Lipid-Rich Plaque Macrophages as Link Between Inflammation and Angiogenesis**

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Background: Macrophages expressing the pro-angiogenic factor VEGF can contribute to plaque progression and vulnerability via induction of intimal neovascularization. Hypoxia Inducible Factor 1-alpha (HIF-1 alpha) has been identified as key transcriptional regulator of cellular VEGF-synthesis and mediates the angiogenic response of tissues to hypoxia. However, little is known about the pattern of expression of HIF-1 alpha in atherosclerosis. Our objective was to assess in parallel the expression of HIF-1 alpha and VEGF in human and experimental atherosclerosis comparing lipid-rich with fibrous plaque.
Methods: In human atheroma (n=12) HIF-1 alpha and VEGF mRNA and protein expression were evaluated by in situ hybridization and immunohistochemistry (data presented as mean±SEM). In NZW-rabbits (n=14) atherosclerotic lesions were induced by aortic double balloon injury and 9 months of high-cholesterol (HC) diet. Five rabbits were sacrificed at 9 months and were used as baseline (BL). The remaining rabbits were then randomized into continued HC diet (n=5) and normal chow diet (ND) (n=4) for another 6 months. Total serum cholesterol at the end of ND treatment was 27±10 mg/dl vs. 861±143 mg/dl at BL (P<.01) and 526±108 mg/dl with continued HC-diet at 15 months (P<.01).
Results: In human atheroma, HIF-1 alpha and VEGF mRNA and protein expression were increased in lipid-rich compared to fibrous plaque (23±5 % vs. 5±2 %, P<.01; 32±6 % vs. 8±3 %, P<.05) and correlated with high macrophage and neovessel content (P<.05). Double-labeling demonstrated co-localization of HIF-1 alpha and VEGF in inflammatory cells surrounding intimal neovessels in close proximity to cholesterol clefts. In plaques of rabbits with continued HC diet and high serum lipid levels expression of both HIF-1 alpha and VEGF-protein as assessed by immunohistochemistry and Western Blot were significantly increased compared to BL and ND.
Conclusions: The selective detection of HIF-1 alpha in VEGF-expressing macrophages located in lipid-rich plaque tissue points to hypoxia as a possible link between inflammation and angiogenesis. Both processes might synergistically contribute to plaque progression and vulnerability.

1045-198 **Influence of Diabetes Mellitus on Coronary Collateral Flow: A Definite Answer to a Rather Elderly Controversy**

Rainer Zbinden, Stefan Zbinden, Michael Billinger, Stefan Windecker, Bernhard Meier, Christian Seiler, Cardiovascular Center Bern, Bern, Switzerland

Background: The influence of diabetes mellitus (DM) on coronary collateral flow is controversial. This may be due to the use of inaccurate means of measuring collateral flow (i.e. angiographic assessment), another reason relates to differences in parameters influencing collateral flow between diabetic and nondiabetic patients (serum lipids, severity of coronary artery disease (CAD)). The purpose of this study was to determine the influence of DM on coronary collateral flow in a large population with variable degrees of CAD using accurate means of collateral flow measurement.
Methods: 187 patients (age 63 +/- 9 years; 86 diabetic and 101 nondiabetic patients) were included in the study. Coronary collateral flow was assessed in 196 stenotic and in 38 angiographically normal vessels using a pressure guide wire (n=145), Doppler guide wire (n=53) or both (n=36) to calculate pressure- or flow-velocity-derived collateral flow index (CFI). Diabetic patients were matched for gender, age, serum lipids and percent diameter stenosis of the vessel undergoing CFI measurement with a nondiabetic control group.
Results: The two groups were exactly balanced for clinical and angiographic data. There was no difference between CFI in the diabetic versus the non-diabetic patients (0.205 ± 0.119 and 0.217 ± 0.128; p=NS). There was still no difference in CFI when only angiographically normal vessels (0.193 ± 0.084 and 0.201 ± 0.072; p=NS) or chronic total coronary occlusions (0.314 ± 0.151 and 0.323 ± 0.186; p=NS) were compared. There was a trend towards a lower number of patients having angina pectoris during the one-minute vessel occlusion in the diabetic group (p = 0.1). There was no correlation between the CFI and the level of HbA1c (as marker for blood glucose control over the last 2-3 months).
Conclusion: There is no difference in coronary collateral flow between diabetic and non-diabetic patients. There is no inverse "dose-response" relation between blood glucose control (represented by the HbA1c level) and coronary collateral flow. There is a trend that diabetic patients have less angina pectoris during a one minute balloon occlusion than non-diabetic patients.

POSTER SESSION

1046

Aortic, Renal, and Peripheral Arterial Diseases

Sunday, March 07, 2004, 3:00 p.m.-5:00 p.m.
Morial Convention Center, Hall G
Presentation Hour: 3:00 p.m.-4:00 p.m.

1046-165 **Measurement of Resting Ankle Brachial Index May Not Accurately Identify Disease in Patients With Signs or Symptoms of Peripheral Arterial Disease**

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Background: The ankle-brachial index (ABI) is useful for objective assessment of lower extremity arterial perfusion, correlating with symptoms and functional severity of peripheral arterial disease (PAD) in population-based studies. This simple, inexpensive measurement can be performed in the primary care office for detection of PAD, and is a marker of increased cardiovascular risk in both symptomatic and asymptomatic patients. The diagnostic accuracy of the resting ABI in patients with symptoms or signs of PAD, however, has not been determined. **Methods:** In order to determine the added diagnostic utility of measuring ABI and pulse volume waveforms (PVR) at rest and after exercise, we analyzed the results of 142 consecutive patients who were referred to the vascular laboratory by *non-vascular* specialists between February and September 2003. **Results:** The average patient age was 68 years; 54% were male. Resting ABI was normal in 44%, abnormal in 38% and not diagnostic due to arterial calcification in 17%. Forty percent were abnormal in those with claudication, 45% in those with ulceration and 28% in those with rest pain. Thirty-six (57%) patients with a normal ABI underwent treadmill exercise testing and 28% of these were found to have an abnormal exercise study indicating the presence of PAD. Among the 24 patients (17%) with calcified vessels, 5 (21%) had abnormal PVR consistent with PAD. **Conclusion:** In symptomatic patients with normal resting ABI, PAD was diagnosed frequently by measuring ABI after exercise. Of the 17% in whom arterial calcification precluded accurate ABI measurement, PVR disclosed PAD in 21%. In patients with suspected PAD, referral to a vascular laboratory for exercise testing and PVR offers more precise diagnosis than office measurements of ABI.

1046-166 **Is the Application of Collagen-Based Vascular Closure Devices After Percutaneous Coronary Interventions Safe and Effective? Intermediate Results From the Prospective Bad Berka Registry**

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Background: Several vascular closure devices (VCD) were developed as an alternative for manual compression after percutaneous interventions (PCI) of the coronary arteries to reduce the time to discharge. We report about the results of the collagen based VCD VasoSeal®ES™ (Datascope Corp.) [VS] and Angio-Seal® (St. Jude Medical) [AS].
Methods: From 10/01 to 09/02 patients with collagen based VCD after percutaneous interventions were enrolled in our prospective registry. During the intervention heparin, aspirin and if necessary clopidogrel were administered. The two VCD were used according to a standardized method, the choice of the device after PCI was left to the discretion of the investigator. Secondary haemorrhage, haematomas (> 5 cm diameter), pseudoaneurysms and AV fistulae were noted as complications. A clinical examination and a Duplex examination in addition was performed routinely at every patient.
Results: 5717 cardiac catheterisations were done until deadline 31.09.2002.
In 1170 cases a VCD was used after coronary intervention, 700 times AS and 470 times VS. One of the above described complications appeared at 54 of 700 cases (7.71%) in the AS group, in the VS group in 35 of 470 cases (7.44 %.) The predominant number of the complications fell on minor complications such as haematoma and smaller secondary haemorrhages (49 of 54 at AS group (7%) and 30 of 35 at VS group (6,4%). Only 5 of 54 (0,7%) of AS and in 5 of 35 (1%) of VS developed more serious complications. One patient in the AS group developed an AV fistula, in four cases we saw pseudoaneurysms. 5 cases of the VS group developed pseudoaneurysms. All aneurysms could be successfully compressed in an ultrasound guided procedure. In one patient of the VS group the